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Correspondence

Acute Exercise and Markers of Endothelial Injury

Sir

The recent study by Woodburn *et al.*[1] found no evidence of vascular endothelial damage following treadmill exercise in claudicants. While the authors' comments may be correct, we doubt that the study they present could in any way lend support to their conclusions. A number of groups have observed evidence of an inflammatory response after maximal exercise in claudicants. Whether this is of short or long-term clinical significance is unclear.

The authors report the results in a small number of patients, some of whom did not walk to their maximum capacity. The timing of the post-exercise sample was presumably based on the authors' previous work following angioplasty. Several clinical studies have used the treadmill model of claudication to define the time-scale of this inflammatory response. Maximal exercise followed by rest leads to free radical formation at 1 min,[2] neutrophil activation at 5 min,[3] neutrophil thromboxane production at 10 min, peaking at 60 min[3] and endothelial damage (urinary microalbumin excretion) at 60 min.[4,5] One would therefore expect an increase in vWF to occur much later than 2 min post-exercise. In fact, Edwards *et al.*[6] reported a significant increase in vWF after 60 min rest in claudicants, with no change in control subjects.

The authors report the effects of a single episode of walking and suggest that this has implications for the role of therapeutic exercise programmes in claudication, which are generally continued for 6 months or more. In fact, we found that regular exercise attenuates this inflammatory response over a period of time.[5]

The exercise-induced inflammatory response in claudicants is well reported. The significance, if any, of the phenomenon is unclear but this paper has done little to contribute to our understanding of this.

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Exercise in Patients with Intermittent Claudication

Sir,

The paper by Woodburn *et al.* will certainly stimulate discussion among clinicians and scientists interested in the effects of exercise in patients with intermittent claudication (IC).

Endothelial injury may lead to progression of POAD or acceleration of coronary/carotid or cerebral artery disease and, is therefore considered important in this patient group who are known to have an increased morbidity and mortality mainly due to adverse cardiac or cerebral events.[1] Endothelial behaviour is, however, only one aspect of a hierarchy of cellular and chemical interactions, also involving platelets neutrophils and cytokines, that occur when patients with IC exercise.

The authors omitted to mention that von Willebrand's factor and thrombomodulin have previously been measured as markers of endothelial damage in patients with IC and serum levels have been shown to rise significantly following exercise,[2,3] but

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